EPIDEMIOLOGICAL CONSIDERATIONS RELATIVE TO SULFUR OXIDES AND PARTICULATES*

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JOHN Knelson's remarks represent one of the most succinct and cogent appraisals of the problem that we have had the fortune to hear in a long time, and it put the matter in better perspective. It is refreshing to hear the admission that perhaps we may be back at square one, and, after listening to Dr. Carl Shy, perhaps we are even further back than that.

As you know, our group has had intimate association with Environmental Protection Agency (EPA) data over the last few years. It has been exciting to reexamine these data for a number of reasons: it has been educational and it gives us an opportunity to report some of our experiences, and in so doing perhaps to help point the way toward the kind of health intelligence which is necessary, not only to establish rational control strategies and standards, but to protect public health, which is really what we are all here for.

Deprecation of any single study will not in itself deprecate an entire program. The CHESS studies and the program as a whole were indeed quite ambitious, but within the framework of these studies there may still lie some pearls for us.

It has been argued that chronic bronchitis or chronic respiratory disease studies show consistent associations that implicate sulfur oxides and particulates. Our analysis of these studies is by no means complete, but we have found a number of major discrepancies. For example, there are problems with the interpretation of results from community to community when they are treated wholly or individually. Rates of questionnaire return, while not extremely low, are inconsistent as can be seen from the

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raw data or draft documents. It is well known that self-administered questionnaires lead to bias problems difficult to quantify and which render the data equivocal relative to yielding the thresholds which we seek.

Further, with few exceptions, actual measurements of air quality are not consistently available and the epidemiologist or the statistician is forced to reconstruct exposure using models which in and of themselves have sources of error, sometimes twofold. This forces attempts to go backward with invalid modeling and similarly establish thresholds using data from studies with high attrition rates and unquantifiable questionnaire problems.

This issue is further confounded when an attempt is made to follow up these studies a few years later and it is found that results are often juxtaposed or contradictory. For example, a very early study of chronic bronchitis in New York City[1] (Dr. Knelson referred to it) stated quite emphatically—and exclusive of the threshold considerations—that sulfates and specifically sulfur dioxides and particulates were responsible for increasing the prevalence of chronic bronchitis in the three New York communities of the Bronx, Queens, and Riverhead. A follow-up study found not only that the previous results could not be confirmed, but that the prevalence rates had reversed themselves.[2] The initial study found that Riverhead, New York, the community which had the lowest pollution level overall had the lowest prevalence rate of bronchitis. By contrast, the high-pollution community of the Bronx had four times the rate. Based on this first study, it would appear that pollution, in this case SO_2 and particulates, was a major determinant in the prevalence of chronic bronchitis. When one contrasts data from 1970 to results obtained in 1972 (this latter data being available only in draft form and not in the form of a formal publication) the overall rates of chronic bronchitis dropped very dramatically. Further, the most drastic decrease occurred in the most polluted community, namely, the Bronx. Given that the second study repeated the first, this observation is difficult to explain. If one accepts the conventional hypothesis that many years of retrospective exposure is a main determinant in increased prevalence of chronic bronchitis, one would not expect so drastic a change within so short a time frame.

These anomalies do not invalidate the hypothesis that there may be some relation between air pollution and chronic bronchitis but illustrate that even with techniques available to us, we are often unable to sort out even the most seemingly straightforward problems, such as the assessment of the prevalence of respiratory disease. The most outstanding anomaly of this

series of studies was that in the first year of study sulfates alleged to be formed by the interaction of SO_2 and total suspended particulates (TSP) in the environment appeared to have increased the prevalence of chronic bronchitis,[1] whereas in the second year sulfates could not be clearly implicated.[2]

That is perplexing. It begins to touch on the specificity of an indicator of disease. One can only surmise regardless of whether pollutant levels change, from year to year, populations change or, unexplainedly their responses to individual pollutants change.

We also reanalyzed two years of CHESS information relative to asthma in New York.[3,4] In the first year sulfates were clearly implicated in the axacerbation of asthma, and adverse-affects thresholds were promulgated.

We reanalyzed that data and found that not only could we not show a relation between aggravation of asthma and sulfates, but more often than not the association between SO_2 and asthma was negative. The CHESS authors had originally found a statistically significant correlation between sulfates and increased asthma attacks, and stated that moreover it was temperature-specific.[3] The next year a follow-up study was performed[4] in which nitrates rather than sulfates appeared to be implicated. I say that nitrates appeared to be implicated because, once again, it is difficult to interpret the conclusions in the draft. We examined these data again, hoping to find an association between increased asthma attacks and some air pollutant. We could neither confirm the nitrate effect nor show any quantitative associations. However, there appears to be a qualitative association between particulate levels and aggravation of asthma.

Thus, our experience with asthma data in New York confirms what Dr. John Knelson stated and perhaps relates to the issues that Dr. Shy mentioned, that is, that the data are not amenable to the kind of quantitative restraints that we put upon it.

Asthma data collected in Salt Lake City have been subjected to very intense analysis.[5] It would appear that it might confirm a relation between higher oxides of sulfur or SO_2 and TSP in combination with the increased exacerbation of asthma. We have not seen the analysis of the data other than the conclusions. However, the data have been further scrutinized by the EPA and by the affected industry itself. This further scrutiny has failed to exonerate SO_2 from a role in aggravation of asthma. Salt Lake City represents a unique situation: it is a community whose pollutant profile is largely dominated by a single very large point source and lends itself to the

drawing of much cleaner pollutant gradients than typically are seen in very large cities. At the very least the results give us a point of reference.

The question of synergy is a challenging hypothesis. Some data were collected in Los Angeles by the CHESS group to assess the effect of simultaneous exposures to sulfur dioxide and oxidant on asthmatics, with the expressed hypothesis that because there is a homogeneous reaction in the South Coast air basin which should drive SO_2 to sulfates there might be some synergism.[6] Since this data base provided a unique scenario in which to study this phenomenon, we performed a reanalysis in conjunction with Southern California Edison personnel.[7] Unfortunately, insufficient data were available to implicate any specific air pollutant in the exacerbation of asthma.

As expected, there were many problems with the study. The original group of asthmatics surveyed totaled about 512, but by the time the sample actually got analyzed, it was reduced to about 327. Strong trends unrelated to pollution appeared in the data. For example, we observed clusters of attacks occurring on Fridays and Saturdays. Other variables, such as pollen, occupational exposures, food allergies, etc., so highly confounded the data that we began to believe that perhaps the data were not really worth analyzing. However, since there was the allegation that it showed synergy, we felt obliged to delve further.

One of the major problems was inaccurate or missing data, particularly with aerometric data which disappear and then reappear. In general, their etiology is obscure. This is not unique to CHESS studies, and occurs with all studies of this type. Often we find the substitution of "appropriate numbers" which are difficult to validate because the data are extrapolated. Many days are deleted from the study because of such difficulties with the aerometrics.

Therefore, it is not surprising that today we hear from this forum that data of this type are not amenable to quantification. As far as we can tell, present data merely suggest that some higher sulfur oxide or congener thereof, or some covarying pollutant, might affect morbidity. What it is, at what concentration, over what sampling time, or what subset of the population is involved, is really not known at this time. There is no really good evidence to implicate clearly any specific sulfur oxide per se, and it will probably take a very long time and much more enlightened studies to obtain the ultimate answer.

Our experience indicates quite clearly that we are almost a light year

away from this goal and as usual we are still trying to design the definitive experiments that will begin to give us even qualitative insights relative to morbidity.

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